

## Acute fatty liver of pregnancy in postpartum - an uncommon time of presentation

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### Abstract

Acute fatty liver of pregnancy (AFLP) is an obstetric emergency can result in maternal and fetal complications including death. The exact pathology of disease is unknown but abnormality in the fatty acid metabolism play an important role. The disease usually affects in third trimester and immediate postpartum(1). The diagnosis of AFLP usually based on clinical presentation and compatible laboratory finding. Intensive care unit admission with supportive management and immediate delivery irrespective of gestational age are main stay of treatment(2). We report a case of 26-year-old mother who was diagnosed with AFLP in second day of postpartum.

### Key words

Acute fatty liver of pregnancy, postpartum, fatty acid

### Introduction

Acute fatty liver of pregnancy first reported in 1940 by sheehan. It is a rare disease and exact pathogenesis is not known (3). The risk factors that play a role are fetal long chain 3-hydroxyacyl CoA dehydrogenase deficiency, prior episode of AFLP, multiple gestation, pre eclampsia or hemolysis, elevated liver enzymes and a low platelet count syndrome, male faetal sex and low body mass index.

### Case presentation

A 26-year-old mother in her third pregnancy with previous two uncomplicated pregnancies was admitted at period of amenorrhea of 37+4 weeks with the complaint of abdominal pain, nausea and malaise of one day duration. She didn't have

vaginal discharge or any bleeding manifestation.

On examination she had pain, mild palor and had muddy sclera. Her pulse rate was 94 per minute, blood pressure was 130/80mmHg Vaginal examination revealed cervical dilatation of 5 cm, 80% of effacement and intact membrane

Her delivery was augmented by artificial rupture of membrane and syntocinon. A healthy baby boy was delivered. Her estimated blood loss during delivery was around 1.5 liter which was attributed to vaginal tear.

Her vitals were persistently normal other than tachycardia. Her hemoglobin was 8 g per dl and platelet was 160000/cumm. She recived one pint of crossmatched blood.

On postpartum day one patient complained generalized itching and persistent nausea.

On postpartum day two patient became drowsy and couldn't cope with the baby. Her GCS was 14. She was deeply icteric and had bilateral flaps. She had distended abdomen with free fluid. Her pulse rate was 110 per minute and blood pressure was normal. Respiratory system and rest of the neurological examination was normal.

Detail investigations carried out in the mother. Complete blood counts showed a hemoglobin: 9.5g/dl, white blood cell count: 8,400/cumm, and platelet count: 57,000/cumm. Liver function tests revealed aspartate aminotransferase: 210 U/l, alanine aminotransferase: 456 U/l, total bilirubin: 95 micromole/dl, direct bilirubin: 66 micromole/dl, alkaline phosphatase: 480 U/l, total protein: 6.8g/dl, and albumin: 2.8 g/dl. Blood picture was leucoerythroblastic and lactate dehydrogenase level

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1125U/l. Biochemical tests revealed blood urea: 10 mmol/dl, serum creatinine: 217 micromole/dl, serum glucose: 80 mg/dl. Coagulation profile revealed a prothrombin time of 24.1 seconds and international normalized ratio (INR) of 2.2. Urine full report showed red cells in urine. Ultra sound scan revealed bright liver suggestive of acute fatty liver of pregnancy. A diagnosis of Acute fatty liver of pregnancy was made.

Patient was transferred to intensive care unit. Vital signs were monitored continuously. Her urine output was on higher side nearly 70-80 ml/hour. Full blood count, liver, renal function test and coagulation profile were done six hourly. Mother had persistent vaginal bleeding and platelet count dropped to 10000/cumm. Her Liver enzymes were persistently high and INR went up to 3. Repeat blood picture revealed persistent hemolysis. Her fluid balance was maintained according to central venous pressure and hypoglycaemia was corrected by 50% dextrose. Liver failure regime was started with lactulose, vitamin K and broad-spectrum antibiotics. Coagulation defects corrected with 4 units of Fresh frozen plasma, 2 units of blood, 4 units of platelets and 7 units of cryo were given. N acetyl cystine infusion was started as per gastroenterologist opinion

After three days in ICU she completely recovered from disease and blood investigations returned to normal. Interval laparoscopic ligation and resection of tubes was done after a month from discharge.

## Discussion

There are many causes Jaundice and bleeding during pregnancy which are AFLP, pre-eclampsia, HELLP syndrome, and viral hepatitis(4,5). Acute viral hepatitis in pregnancy is usually presents as a systemic illness with jaundice, fever, nausea, vomiting and fatigue. Liver enzyme elevation is more marked in viral hepatitis and its usually more than five hundred. Other three condition, AFLP, pre-eclampsia, HELLP syndrome may coexist and it difficult to differentiate from each other.

Gestational age 30 weeks to 38 weeks is a typical time of presentation of AFLP but diagnosis can be early as 22 weeks and late as four days after delivery.

In our patient we diagnose the case in postpartum day two. Acute fatty liver patients' initial symptoms are often nonspecific which include nausea, vomiting, abdominal pain, malaise, headache and anorexia(4). In advanced disease patient may have features of acute liver failure, including jaundice, ascites, encephalopathy, disseminated intravascular coagulopathy, hypoglycemia, features acute kidney injury and eventually multi organ failure(6)

These patients can have polyuria like our patient. This is mainly because of central diabetes insipidus. It is caused by decreased arginine vasopressin secondary to reduced clearance of vasopressinase by impaired liver

The diagnosis of AFLP supported by Swansea criteria which include vomiting, abdominal pain, polyuria, encephalopathy, elevated bilirubin greater than 14 micromole/l, hypoglycemia less than 73mg/dl, leukocytosis greater than 11000cells/microl, elevated transaminases more than 42IU/l, elevated ammonia greater than 47 micromol/l, elevated uric acid more than 340 micromole/l, acute kidney injury or creatinine more than 1.7 mg/dl, coagulopathy or prothrombin time greater than 14 seconds, ascites or bright liver in ultrasound scan and microvascular steatosis on liver biopsy. At least 6 criteria need to be positive to diagnose AFLP.

Management of AFLP includes of rapid delivery of the fetus and supportive treatment. Most of the complications will resolve with in two to three days of delivery. Fluid replacement should be guided by central venous pressure. Over enthusiastic fluid therapy will result in complication due to overload. Plasma exchange plays a role in very severe cases which are not respond to routine measures. It helps to clear the harmful metabolites and improves the disease. In Gestational age less than 32 weeks mothers, Magnesium sulphate is reducing the risk of cerebral palsy and severe motor dysfunction in fetus. There is a chance of recurrence in subsequent pregnancies. so following pregnancies should be managed with maternal faetal medicine specialties

## Conclusion

Acute fatty liver of pregnancy usually affects in third trimester and immediate postpartum of pregnancy. Initial presentations commonly with vague symptoms. Prompt diagnosis, early delivery and vigilant supportive management is lifesaving. Mothers who developed complications should admitted to Intensive care unit and managed by multidisciplinary team.

## References

1. Riely CA. Liver disease in the pregnant patient. *Am J Gastroenterol.* 1999;94:1728–32.
2. Usta IM, Barton JR, Amon EA, Gonzalez A, Sibai BM. Acute fatty liver of pregnancy: An experience in the diagnosis and management of fourteen cases. *Am J Obstet Gynecol.* 1994;171:1342–7.
3. Riely CA. Acute fatty liver of pregnancy. *Semin Liver Dis.* 1987;7(1):47–54. doi: 10.1055/s-2008-1040563.
4. Wei Q, Zhang L, Liu X. Clinical diagnosis and treatment of acute fatty liver of pregnancy: a literature review and 11 new cases. *J Obstet Gynaecol Res.* 2010;36(4):751–756. doi: 10.1111/j.1447-0756.2010.01242.x.
5. Pang WW, Lei CH, Chang DP, Yang TF, Chung YT, Huang MH. Acute jaundice in pregnancy: acute fatty liver or acute viral hepatitis? *Acta Anaesthesiol Sin.* 1999;37(3):167–170.
6. Knight M, Nelson-Piercy C, Kurinczuk JJ, Spark P, Brocklehurst P. UK obstetric surveillance system: a prospective national study of acute fatty liver of pregnancy in the UK. *Gut.* 2008;57:951–956. doi: 10.1136/gut.2008.148676.